

Section of Neurology

President—GEOFFREY JEFFERSON, M.S.

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The Radiological Diagnosis of Chronic Subdural Hæmatoma

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THE primary object of this communication is to attempt to demonstrate the value of neuroradiology in the elucidation of the diagnosis of subdural hæmatoma. The condition has been recognized for many years, and is now becoming more frequently diagnosed.

History.—The earliest case I have been able to trace was described by Burrows in the Croonian Lectures for 1835. A man, aged 53, was admitted to St. Bartholomew's Hospital in October 1834, under Dr. Latham.

"The man stated that his mind had been a good deal oppressed for a twelvemonth, in consequence of adversity, and that six months prior to his admission he had been attacked with headaches which were relieved by blisters. About a month before admission, headache and giddiness returned which prevented him from following his occupation. Upon his admission he complained of giddiness with inability to retain his urine or fæces. He was cupped from the temples and blistered; but his mental faculties gradually declined and he remained in a state of insensibility approaching to coma; without any material change he lingered a few days and died."

The post-mortem examination is carefully described and the lesion was clearly a chronic subdural hæmatoma. Burrows said: "The left hemisphere of the brain was much compressed by the distended sac of blood and coagula; the left ventricle was small from pressure; there was nothing further remarkable in the substance of the brain." The specimen was placed in the St. Bartholomew's Hospital Museum.

In 1845 at this Society Sir Prescott Hewitt, the Curator at St. George's Hospital, delivered a masterly account of the lesion. He described what is probably the first published bilateral case. It was in a man aged 51, with a history similar to that of Burrows' case. In neither author's material was trauma mentioned in the history and no fracture of the skull was found at autopsy.

It would not perhaps be an altogether idle speculation to suggest that had not Sir Benjamin Brodie shown so much reluctance about opening the dura, for he found that all such cases went septic afterwards, he might well have been the first to cure cases of subdural hæmatoma. He was after all a colleague of Sir Prescott Hewitt's, and was well versed in the science of the cranial surgery of the time.

In 1905 Bowen made an important contribution, collecting 72 cases from the literature including one of his own.

In 1914 Trotter described four cases upon which he had operated. He was perhaps the first of the more modern authors to stress the importance of the disease. He stated that the lesion probably frequently escaped clinical recognition, and he made a plea for establishing the diagnosis in the early stages of the disease, for he said cure would then result in every case.

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Recently papers have appeared in the literature describing quite a large series of cases. Nearly all these are from the U.S.A. Munro (1934) describes 62, Kunkel and Dandy (1939) 48, Dyke and Davidoff (1938) 24, and Munro and Merritt (1936) reviewed the surgical pathology of 105 confirmed cases. But the largest series of all was that of Allen, Daly and Moore (1935), who described 245 cases out of a material of 3,100 autopsies on patients from mental hospitals. This represents 7·9%. Holt and Pearson (1937) describe three cases from mental hospitals which were diagnosed by encephalography. Two of these three cases were cured both mentally and physically, and the third developed an osteomyelitis of the finger which ultimately led to his death. These three cases all had bilateral hæmatomas.

Munro, an acknowledged expert on head injuries, states that it is regrettable that when car accidents are becoming increasingly more common and thus increasing the toll of head injuries, the lesion is not more frequently diagnosed. The figures of Allen, Daly and Moore, make it quite clear that mental patients should be investigated more carefully with subdural hæmatoma in mind.

Ætiology.—Subdural hæmatoma is far more common in men than in women. Various ratios have been given by different authors, but all are agreed as to the great predominance in men. The condition may occur at any age from intra-uterine life upwards. In a large percentage of cases there is a history of trauma, either recent or remote. But most observers are agreed that a not inconsiderable proportion of the cases give no history of trauma at all. In Kunkel and Dandy's series 39·6% gave no history of trauma and only one out of the 48 cases showed a fracture of the skull. None of Dyke and Davidoff's showed fracture. In the present series of 30 cases (less four children to be described elsewhere) 11 of the remaining 26, or 42·3%, gave no history of trauma, and in no case was there evidence of a fracture of the skull on X-ray. Even allowing for the fact that many such patients are poor witnesses, for a number of them (9 out of 26) showed some mental changes, it is likely, considering also the absence of fracture of the skull, that any gross trauma, sufficient for the patient to remember, is not an essential factor in the ætiology.

Leary (1934) stresses the importance of alcoholism, which he found a common feature in his cases. He states that it probably acts in two ways :—

(1) By producing an œdema of the arachnoid, and thus serving mechanically to favour rupture of bridging veins, and (2) alcoholics are more addicted to injury than normal individuals.

Leary also found significant subdural hæmatomas in 10% of a series of several hundred fatal traumatic cases.

Pathology.—It is generally accepted that the condition is initiated by a tearing or rupture of the small veins traversing from the arachnoid to the dura. This laceration in more than half the cases follows trauma. In the remaining cases there may be a predisposing pathological lesion of the veins. In some cases it is possible that a gross rise in venous pressure, relative to the pressure in the subdural space, would allow a spontaneous rupture in an otherwise healthy vein or venule. But at the moment this must remain a conjecture. The bleeding presumably continues until the venous pressure is overcome by the general subdural pressure. As with hæmatomas elsewhere in the body a reaction of fibroblasts occurs at the periphery, and a capsule is ultimately formed. The blood may or may not remain fluid, and later, in rare cases, the hæmatoma calcifies.

Dyke and Davidoff maintain that a space exists between the hæmatoma and the dura, on the one hand, and the arachnoid on the other. This, they say, is a pathognomonic radiological sign. We find this view difficult to accept owing to the expanding nature of the process, and the somewhat raised intracranial pressure usually associated. Moreover, we have not been able to find the sign in any of the present series of cases. The situation of the hæmatoma is nearly always on the lateral convexity of the brain, the medial spread being limited by the falx. Why

this is the site of predilection is as yet unexplained. Possibly it is due to the greater number of veins in that situation which are draining to the superior sagittal sinus. Bilateral cases are not uncommon.

The clinical picture, in common with that of most other expanding processes in the cranium, varies very much. At one end of the scale are those cases which present only mental changes and headache. At the other end are cases with symptoms and signs of gross intracranial pressure. Frequently there are no localizing signs, and even if the correct diagnosis is made clinically, often there is nothing to indicate on which side the lesion is situated.

Material

Table I consists of 30 cases, four of which are dealt with in another paper and are of the infantile type. All the cases are collected from Professor Olivecrona's neuro-surgical clinic in Stockholm. This table shows the years during which the cases

TABLE I.

Year	Number of subdural hæmatomas	Total expanding processes	Percentage subdural hæmatomas
1931	1	89	1.2
1932	1	106	0.9
1933	0	156	0.0
1934	2	172	1.2
1935	2	219	0.9
1936	3	197	1.5
1937	8	226	3.5
1938	7	242	2.9
1939 (to June)	6		

were admitted. Since 1937 these cases represent about 3% of all intracranial expanding processes admitted under Professor Olivecrona, and in 1939 the percentage is likely to be even higher (six cases in the first half of the year).

Table II (pp. 4, 5, 6 and 7) which is after Kunkel and Dandy, but slightly modified, classifies the chief features. It will be noticed that the cases are all males. The oldest was 67 years and the youngest 17 years (two cases). The average age was 44 years, which approximates closely to Kunkel and Dandy's figures (41.2 years), and Dyke and Davidoff's (44.5 years).

Headache was present in every case and it was always the first cerebral symptom. Of those 15 cases in which a history of trauma was elicited, a latent interval occurred in seven (43%). This varied from a few days to three months, and in one case twenty-one years. In such a case it is of course doubtful as to whether the injury was associated with the hæmatoma. In nine cases there was obvious mental confusion, 10 cases suffered from nausea and vomiting, 12 from diplopia, 20 had some degree of papilloedema. Papilloedema was by far the most constant sign. Table II shows how physical examination was almost entirely negative in other respects, and it was usually impossible to localize the side of the lesion.

A confident clinical diagnosis was made in seven cases, and in a further four cases it was put forward as being rather more likely than tumour. In the remaining 15 cases cerebral tumour was diagnosed. It is hoped to show later that radiology leads to a far more accurate diagnosis not only of localization, but also of pathology.

Twelve of the hæmatomas were on the right and twelve on the left, and two were bilateral. Of these cases one was in the left temporal region and one around the pituitary.

Fourteen cases were discharged cured, one of which (No. 1) died five years later of a bronchial carcinoma. (This represents a 54% cure rate.) Three were definitely improved. Five were not fit for any work, and one of these developed a right

TABLE II.—SUMMARY REPORT OF

Patient, No. and date of admission	Age, sex, (all male)	Chief symptoms	Type of trauma before admission	Duration of symptoms	Latent interval	Headache	Nausea or vomiting	Drowsiness	Diplopia	Vertigo	Convulsions	Coma	Mental confusion	Papilloedema	Involvement of other cranial nerves	Motor and sensory changes
1 E. T. 59/31 12.1.31	37	Headache 8 years on and off. Un- conscious once 3 weeks before admission	Nil	8 yr.	Nil	+	-	-	-	-	-	-	-	+	R.V. VII	Nil
2 G. J. 2538/32 10.10.32	17	Headache fol- lowed injury	4 months before admission hit on head by piece of iron	4 mo.	Nil	+	+	-	+	-	+	-	-	+	Nil	Some rigidity of neck
3 K. J. 1679/34 18.6.34	19	Headache 2 months	Nil	2 mo.	Nil	+	+	+	+	-	-	-	-	+	L. VI	Nil
4 L. W. 3191/34 12.11.34	37	Headache	5 months before admission fell on head off bicycle	2 mo.	3 mo.	+	-	+	-	-	-	-	+	-	Nil	Weakness L. arm, L. leg
5 H. P. 1501/35 31.5.35	27	Headache 3 weeks	Nil	3 wk.	Nil	+	-	-	+	-	-	-	-	+	Right ptosis, R. III	Nil
6 P. P. 196/36 2.4.36	50	Headache	4 weeks ago fell on icy steps. No head injury	3 wk.	Few days	+	-	+	-	-	-	-	+	-	Nil	Nil
7 E. A. 269/36 12.5.36	52	Headache	2 months ago fell off bicycle on head	2 mo.	Nil	+	+	+	+	+	-	-	-	+	R. VII	Nil
8 K. P. 404/36 18.7.36	59	Headache 2 weeks (complic- ated by pituitary tumour)	2 weeks ago hit head	2 wk.	Nil	+	-	-	+	-	-	-	-	-	Nil	Nil
9 P. L. 186/37 1.4.37	60	Headache 4 months on and off	Nil	4 mo.	Nil	+	-	-	+	-	-	-	+	-	L. VI	Muscle power lessened
10 J. N. 322/37 4.6.37	55	Headache 4 weeks after injury	8 weeks ago head injury in car	4 wk.	4 wk.	+	-	-	-	+	+	-	+	+	Nil	Nil
11 A. H. 400/37 9.7.37	17	Headache 1 month	Nil	4 wk.	Nil	+	-	-	+	-	-	-	-	+	R. VI	Nil
12 J. H. 458/37 10.8.37	61	Headache 3 months after accident	4 months ago motor-cycle accident: head injury	1 mo.	3 mo.	+	-	-	+	-	-	-	+	+	Nil	Nil
13 K. D. 619/37 27.10.37	26	Mental changes, headache 4 months	Nil	4 mo.	Nil	+	+	+	+	-	-	-	+	+	Nil	Nil
14 G. G. 658/37 16.11.37	28	Headache 1 month	Nil	1 mo.	Nil	+	+	-	-	-	-	-	-	+	Nil	Nil
15 K. A. 740/37 27.12.37	40	Headache 3 years	Nil	3 yr.	Nil	+	+	-	-	+	-	-	-	+	Nil	Nil

26 CASES OF SUBDURAL HÆMATOMA.

Deep reflexes	Clinical Impression T.C. = tumour cerebri, S.D.H. = subdural hæmatoma	Straight X-ray findings	V. = Ventriculography, E. = Encephalography. Diagnosis on burring	Arteriography	Side of lesion	Recurrence after operation	Result	Follow-up
Normal	T.C. Glioma	Nil	V.	No	L.	No	Cured	Died 25.1.36 carcinoma bronchus. Liver and spine metastases
Normal	T.C. brain-stem	Nil	V.	No	L.	No	Cured	Last communication 24.11.36. Very fit
Normal	T.C.	Nil	V.	No	L.	No	Better: slight speech difficulty	28.1.36: Quite well
Left more active than right	Right S.D.H. or T.C. frontal	Pineal calcified normal place	E.	No	R.	No	Cured	7.4.39: Quite well
Normal	T.C. or S.D.H.	Pineal calcified $\frac{1}{2}$ cm. to left	E.	No	R.	No	Cured	14.4.39: Quite well
Bilateral Babinski	S.D.H.	Nil	E. No air in ventricles. Burring	No	L.	No	Cured	8.9.38: Quite well
Right Babinski	Left S.D.H. or T.C. frontal	Pineal calcified normal place	E. No air in vent. Hæmatoma found on burring	No	L.	No	Cured	1.9.37: Quite well
Normal	Pituitary adenoma chromophobe	Enlarged sella	Nil	Yes	R.	No	Improved. Still headache	X-ray treatment. Less well in 1939
Normal	S.D.H.	Nil	E.	No	Bilateral	Unconscious after operation. after (Extradural hæm. —reoperated)	Died 3 days after operation.	
Normal	T.C.	Nil	V. Only contra-lateral ventricle filled	No	L.	No	Cured	22.12.38: Quite well
Normal	T.C.	Not made	Burring	No	R.	No	Cured	31.3.39: Quite well
Normal	S.D.H. or T.C.	Nil	Burring	No	R.	No	23.11.38: Headache now and then; cannot work	
Normal	T.C.	Calcified pineal, normal place	Burring	No	Bilateral	No	Cured	17.4.39: Quite well
Normal	4th vent. tumour	Widened sutures. Dorsum sellæ thin	V.	No	L.	No	Cured	18.3.39: Quite well
Normal	T.C.	Nil	V.	No	R.	No	Operation, good result	29.9.38: In alcoholic home

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16 A. S. 96/38 5.2.38	39	Headache 4 months	5 months ago fell on back of head. Unconscious	4 mo.	1 mo.	+	+	-	+	-	-	-	-	+	Nil	Nil
17 E. P. 390/38 23.5.38	36	2 months epi- lepsy, 1 month headache	Head injury 3 months ago	3 mo.	1 mo.	+	-	-	-	-	+	+	-	+	Nil	Nil
18 J. E. 440/38 13.6.38	67	Headache 2 months	Nil	2 mo.	Nil	+	-	-	-	-	-	-	-	+	Nil	Nil
19 J. A. 490/38 4.7.38	47	Headache fol- lowing injury	Hit head on post 10 days ago. Not unconscious	10 days	Nil	+	+	+	-	-	-	+	-	-	Nil	Nil
20 M. J. 938/38 16.12.38	65	4 months ago spots before eyes, then headache	Nil	4 mo.	Nil	+	-	-	-	-	-	-	-	+	Nil	Muscle weakness, left greater than right. Slight atrophy left side. Deep sensitivity impaired
21 O. L. 83/39 17.1.39	44	4 months head- ache, vomiting, drowsiness	4 months ago hit head against post. (Admitted this after operation)	4 mo.	Nil	+	+	+	-	-	-	-	+	+	Nil	Nil
22 F. E. 160/39 28.2.39	43	Headache and pain in face 21 years. Nervous 2 years	Bullet wound in head 21 years ago. ? type of injury	21 yr.	Nil	+	+	-	-	-	-	-	+	-	Nil	Nil
23 A. F. 205/39 15.3.39	62	Pain and weak- ness R. arm 8 years ago. Dizzi- ness 1 year. Head- ache 1 month	Nil	8 yr.	Nil	+	-	-	-	+	-	-	+	+	Nil	Nil
24 B. R. 286/39 22.4.39	51	Headache 2 months. Dim- ness of vision 1 month	Fell off a bus 2 months ago, unconscious	2 mo.	Nil	+	-	-	-	-	-	-	-	+	Nil	Nil
25 G. B. 299/39 26.4.39	59	Headache 1 month	6-7 years ago struck on head by hammer. No headache	1 mo.	? 6-7 yr.	+	-	-	+	-	-	-	-	+	Nil	Right hand, arm, leg, weak
26 G. G.	51	Headache 1 week	6 weeks ago thrown off a cart. Hit head — un- conscious	1 wk.	5 wk.	+	-	-	+	-	-	+	-	+	Nil	Nil

hemiplegia after operation (Case 21). One case was complicated by a pituitary adenoma and is still receiving deep X-ray treatment (No. 8). Three cases died as the result of operation (11.5% mortality). Case 26 had not left hospital but was an operative success.

Radiology

Plain films.—Of the 26 cases all but two (11 and 25) were examined by “straight” X-ray. Five survey views of the skull were taken in each case, as is the routine custom—postero-anterior, half-axial, axial, right and left lateral (Lysholm, 1931).

26 CASES OF SUBDURAL HÆMATOMA, *continued.*

Deep reflexes	Clinical Impression T.C. = tumour cerebri, S.D.H. = subdural hæmatoma	Straight X-ray findings	V. = Ventriculography, E. = Encephalography. Diagnosis on burring	Arteriography	Side of lesion	Recurrence after operation	Result	Follow-up
Normal	S.D.H.	Thin dorsum sellæ	E. No air in vent. Diagnosis on burring	No	R.	No	Operation, good result	16.2.39: Cannot work. Headache
Normal	S.D.H.	Calcified pineal, normal place	E.	No	L.	No	Nov. 1938 weak left arm—tired—poor memory. Readmission March 1939, wasting L. arm, slight papilloedema. Refused enceph. Discharged	
Normal	T.C.	Calcified pineal, normal place	Burring	No	R.	No	Cured	
Normal	S.D.H.	Calcified pineal, normal place	E.	No	R.	Drowsy following opn. Reoperated. No bleeding found	Died pneumonia 4 days after operation	
Right Babinski	T.C.	Pineal calcified, 5 mm. to left	V.	No	R.	No	Operation, good result	28.4.39: Feels weak, left hand stiff. Cannot work
Right Achilles ++	T.C. malignant glioma	Nil	V.	Yes	L.	No	Drowsiness less but right hemiparesis	
Normal	T.C.	Nil	V.	No	L.	Reoperated for extradural bleeding	Died 12.3.39. Uræmia	
Normal	T.C.	Choroid plexus calcified. No displacement	E.	No	R.	No	4.5.39: Discharged much improved	
Normal	S.D.H.	Nil	E.	No	R.	No	13.5.39: Discharged cured	
Normal	T.C.	Not made	V.	Yes	L.	No	11.5.39: Discharged cured	
Normal	S.D.H.	Nil	E.	No	L.	No	Operation successful. Not followed up	

Findings on Plain Films

- (1) No fracture was seen in any of the cases.
- (2) Evidence of increased intracranial pressure was found in only two cases:—
 - (a) No. 14—widening of sutures of vault.
 - (b) No. 16—thinning of dorsum sellæ.
Increased digitations of the bones of the vault were not observed in any of the cases.
- (3) No gross bone changes or abnormal vascularization of the bones was seen.

(4) The pineal gland was seen to be calcified in eight cases, but only in two cases (Nos. 5, 20) was it displaced laterally.

(5) The choroid plexus was calcified in one case but was not displaced.

(6) Calcification of the hæmatoma was not seen. (Had it been present it would have been pathognomonic—see infantile type.)

(7) Theoretically the shadow of the skull on the affected side should be denser than on the normal side, even in the absence of calcification; for blood contains far more iron than brain tissue and iron is, of course, an element with a high X-ray absorption coefficient. However, I was unable to demonstrate any such finding.

Pneumo-encephalography.—At the Royal Serafimer Hospital encephalography rather than ventriculography is regarded as the method of choice in investigating the brain and intracranial contents, provided that there is no evidence of greatly increased intracranial pressure or a suspicion of a posterior fossa lesion. It has the obvious general advantage of giving information about the convexity of the brain, the basal cisterns, and occasionally the subdural space, as well as the ventricular system. Ventriculography in most cases only gives information about the ventricles. Air injection for encephalography is always performed by suboccipital puncture. Sjöquist (1937) described the method as follows: The patient is placed in the sitting position and the head is supported in front by an assistant. The operator sits behind facing the back of the patient's head. The suboccipital region is shaved and sterilized. The skin and deeper structures are anesthetized with a suitable local anæsthetic. After puncturing the dura the needle is advanced about 2 mm. Using a 10 c.c. syringe 10 c.c. of liquor are removed by suction and an equivalent quantity of air replaced. This technique is repeated until about 30 c.c. of air have been injected. The patient is then sent to the X-ray department on a stretcher trolley.

This method is found to be more satisfactory than lumbar air injection. First, less fluid needs to be removed, for the spinal theca need not be drained and less air need be injected. Secondly, as a result of this the air does not travel up the spinal cord, and thus the cord is not irritated. The symptoms are found to be less irksome to the patient, presumably for this reason. In fact, many patients have no discomfort at all. At the Royal Serafimer Hospital this technique has been employed by the neurologists, neurosurgeons, and psychiatrists for the past ten years, and many thousands of cases have been investigated by this means. Not only has there been not a single fatality, but in expert hands the ventricles have been satisfactorily filled with air in over 90% of cases.

In this material 11 cases were examined by encephalography. Seven of these showed satisfactory filling of the ventricles, and the position of the expanding process could be located (see diagrams).

One of these seven cases proved later to be bilateral (see below). One case showed filling of the contralateral ventricle only. In this case (No. 19) sufficient evidence was obtained to justify a burr hole being made on the opposite side in order to introduce air. On burring the hæmatoma was found. In the remaining three cases no air was seen in the ventricles and ventriculography was next performed, as is the usual custom.

Ventriculography.—It will be noticed that ventriculography was employed more often than encephalography, but even so encephalography has been used relatively more often recently.

In investigating subdural hæmatomas ventriculography has a special advantage in that on making the burr-holes and exposing the dura one may make a certain diagnosis of hæmatoma from the appearances of the dura, and thus air need not be injected at all.

Sixteen cases were examined by this method, including two of the three cases above. The other case was explored on clinical grounds alone. This accounts for all but one of the cases (No. 8), where a pituitary tumour was present, and air examination of the brain was not regarded as being indicated. The subdural

hæmatoma in this case was found by chance, and it was situated in an atypical position (*see later*).

Of these 16 cases six were diagnosed on making the burr holes. Of the remaining 10, satisfactory filling of the lateral ventricles was obtained in nine cases (*see diagrams*). In the tenth case (No. 10) air was present only in the contralateral ventricle. As is customary in such cases a frontal burr hole was made on the affected side in the hope that air could be injected by this means. However, on burring, the dura was found to show the typical appearances of an underlying hæmatoma, and so the air injection was not pursued.

The air-filled encephalograms and ventriculograms will now be considered together, for they show common features. To summarize the material we have:—

Clinical diagnosis	1
Chance finding with pituitary	1
Ventriculogram burr-hole diagnosis	6
<i>Encephalogram—</i>	
Good filling	7
Contralateral ventricle filling	1
(Non-filling .. 3)	
<i>Ventriculogram—</i>	
Good filling	9
Contralateral ventricle filling	1
Total	26

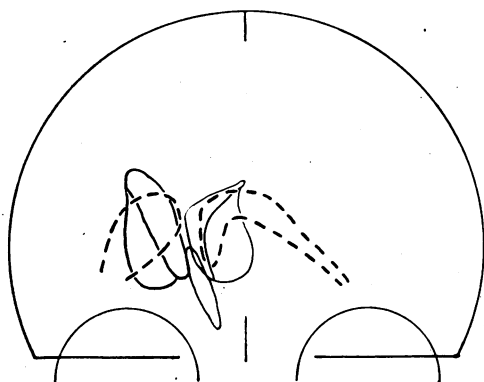


Fig. 1.
CASE 2.

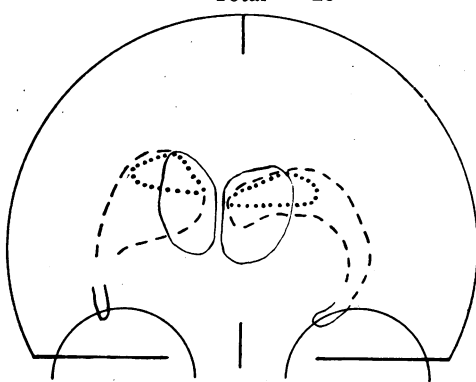


Fig. 2.
CASE 3.

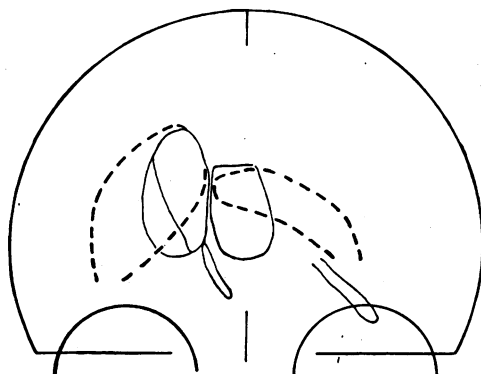


Fig. 3.
CASE 4.

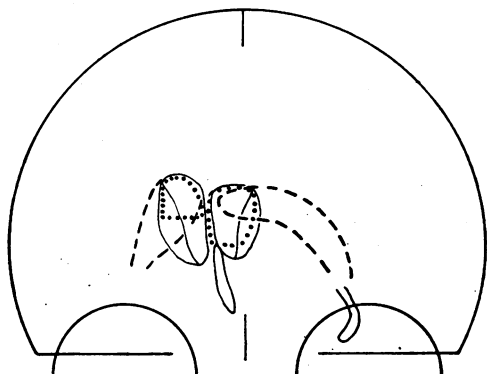


Fig. 4.
CASE 5.

— Pars anterior (patient supine).
 Cella media (patient sitting).
 - - - - - Pars posterior (trigonum) (patient prone).

Of the 18 air-filled cases, 12 showed remarkable similarity (Nos. 2, 3, 4, 5, 14, 15, 20, 21, 23, 24, 25, and 26). Drawings were made of the ventricular systems in all these cases, and for the most part, except for the demonstration of falx pressure, the anteroposterior drawings are the most important. Reduced reproductions of these drawings are shown (figs. 1-12). For the sake of simplicity and diagrammatic

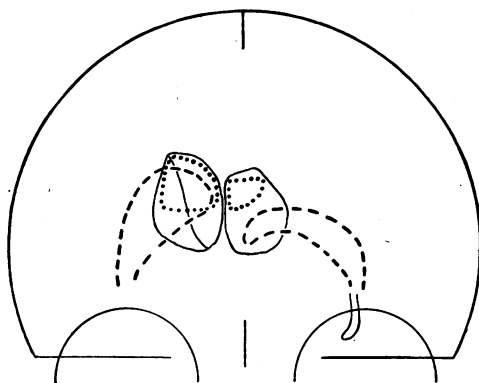


Fig. 5.
CASE 14.

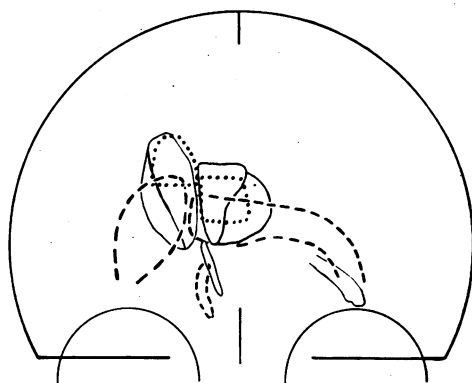


Fig. 6.
CASE 15.

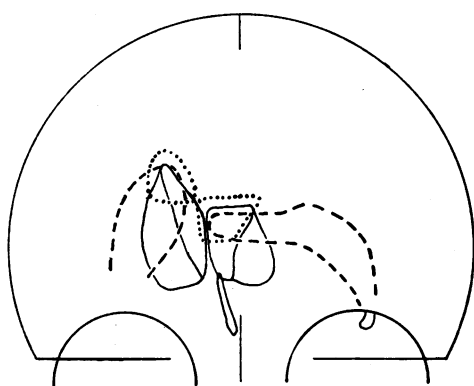


Fig. 7.
CASE 20.

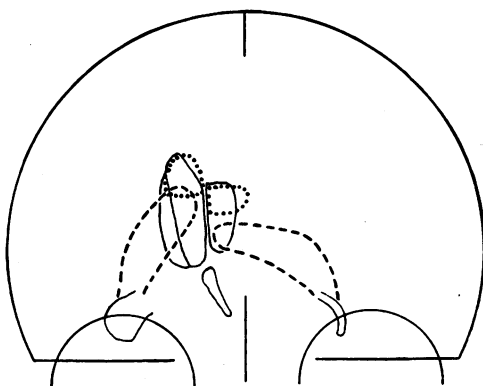


Fig. 8.
CASE 21.

—— Pars anterior (patient supine).
 Cella media (patient sitting).
 - - - - Pars posterior (trigonum) (patient prone).

representation the lesion is represented on the same side in each case. (Fig. 9, Case 23, showed a congenital anomaly—a cyst of the septum pellucidum.)

The features noted are :—

A. Anterior Horns.

- (1) Marked septum pellucidum shift, with negligible angulation.
- (2) Shift, and angulation of the anterior part of the third ventricle with the sagittal plane.
- (3) Angle between septum pellucidum and third ventricle.

- (4) Marked dilatation of contralateral ventricle.
- (5) Very slight or absent dilatation of lateral ventricle on affected side.
- (6) Flattening of roof of lateral ventricle on affected side.
- (7) Elevation of roof on contralateral side.
- (8) Normal position or various grades of medial displacement of temporal horn on affected side.

B. Sitting Position (pars media filled).

- (9) More marked flattening of roof of lateral ventricle on affected side.
- (10) Greater displacement of septum pellucidum.

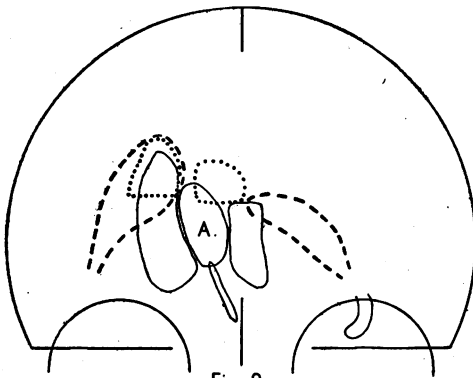


Fig. 9.

CASE 23.

A, Septum pellucidum cyst.

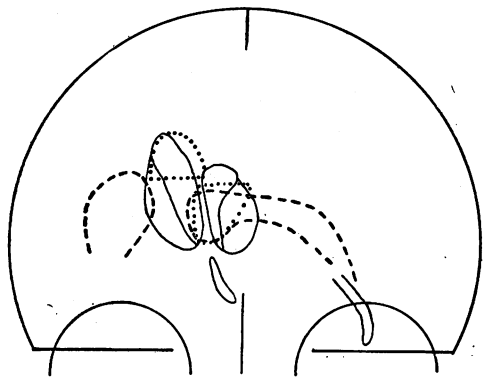


Fig. 10.

CASE 24.

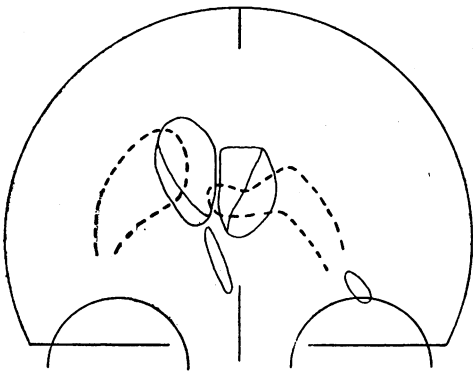


Fig. 11.

CASE 25.

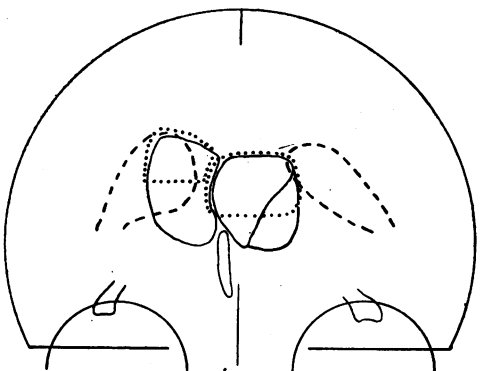


Fig. 12.

CASE 26.

- Pars anterior (patient supine).
 Cella media (patient sitting).
 - - - - - Pars posterior (trigonum) (patient prone).

C. Trigone.

- (11) Flattening of roof.
- (12) Slightly less shift of septum pellucidum than in B (pars media) but greater than A (pars anterior).

Lateral View.

Evidence of falx pressure in cases of great shift of septum pellucidum.

Superimposed drawings of these 12 cases were made, and it was found that they corresponded very closely. However, the result was so complicated in appearance that a synthetic drawing of the "average" was compounded in the three positions: A. Supine (pars anterior); B. Sitting (pars media); and C. Prone (pars posterior)

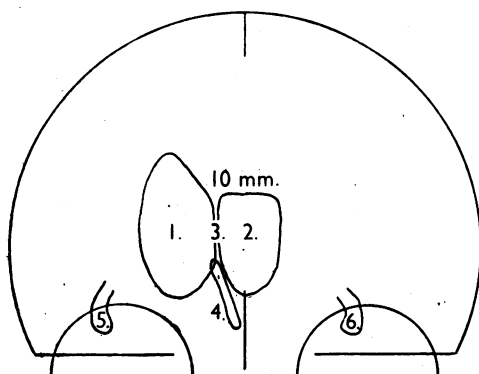


Fig. 13.

Mean drawing in supine position. Note: 10 mm. shift of septum. Medial displacement of 6.

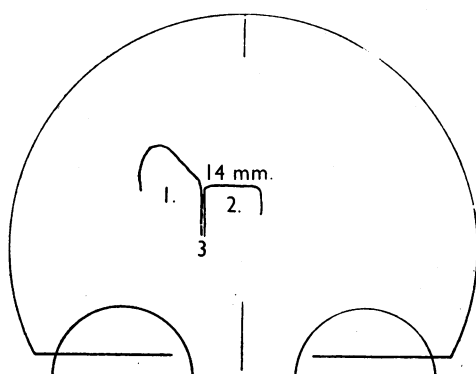


Fig. 14.

Mean drawing in sitting position. Note: 14 mm. shift of septum.

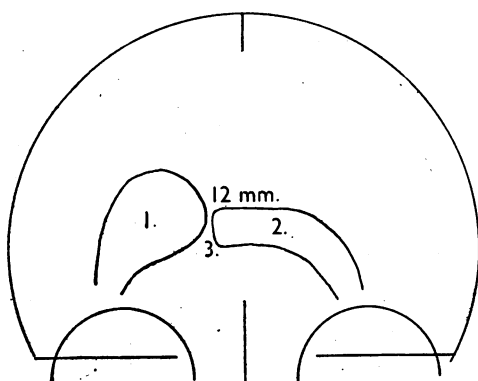


Fig. 15.

Mean drawing in prone position. Note: 12 mm. shift of septum.

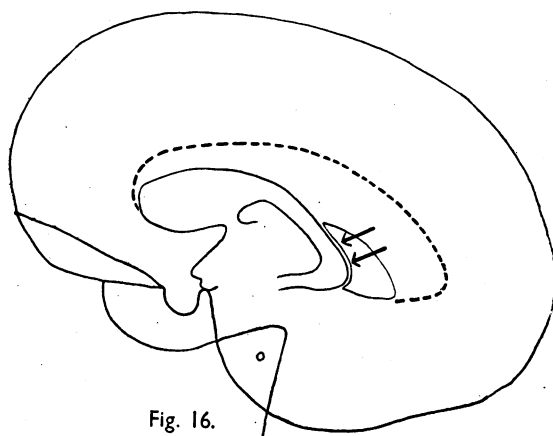


Fig. 16.

Typical lateral drawing (Case 20). Note: Falx pressure; contralateral ventricle dotted.

KEY TO FIGS. 13, 14 AND 15.

1. Contralateral ventricle: dilated, lateral roof high.
2. Homolateral ventricle: not dilated, roof flat.
3. Septum pellucidum: shifted but vertical.
4. Anterior part of third ventricle: upper part shifted laterally; forms angle with septum pellucidum.
5. Contralateral temporal horn: normal position.
6. Homolateral temporal horn: medial shift.

These drawings (figs. 13, 14, 15) show the above tabulated findings more clearly. The mean shift of the septum pellucidum in the three positions was measured and found to be 10 mm. anteriorly, 14 mm. in the cella media, and 12 mm. in the trigone region. One would expect this to be so with a laterally placed lens-shaped expanding

PLATE I.



FIG. 17.



FIG. 18.



FIG. 19.



FIG. 20.—Arrows indicate falx pressure.

FIGS. 17-20 show radiographs taken in positions shown in figs. 13-16.

PLATE II.



FIG. 21.—The foramina spinosa are circled.

process. Fig. 16 shows a typical lateral drawing. Figs. 17-20 show radiographs taken in positions shown in figs. 13-16.

The greater or lesser degree of the medial shift of the temporal horn depends upon the degree of spread of the hæmatoma into the temporal fossa.

This feature, in combination with the others, is regarded by us as being the

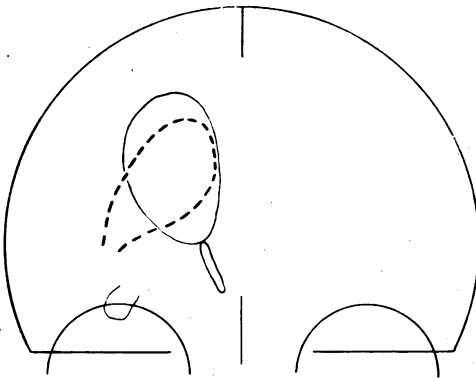


Fig. 22.

CASE 19.—Filling of contralateral and third ventricles only.

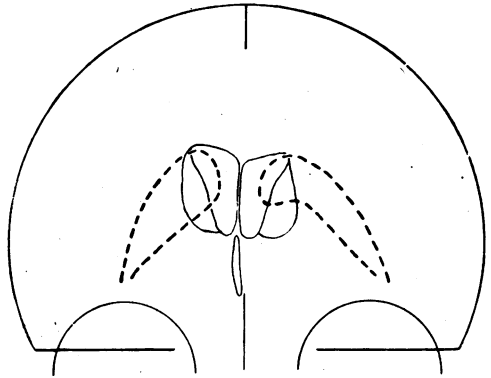


Fig. 23.

CASE 17.—Very slight lateral shift; no classical characteristics.

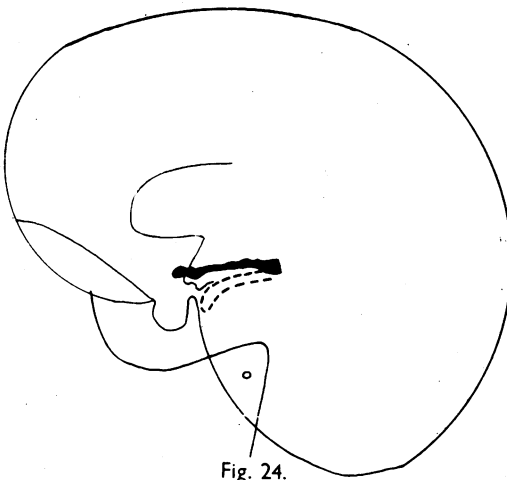


Fig. 24.

CASE 22.—Temporal horn in black; elevated and ragged. Normal situation dotted.

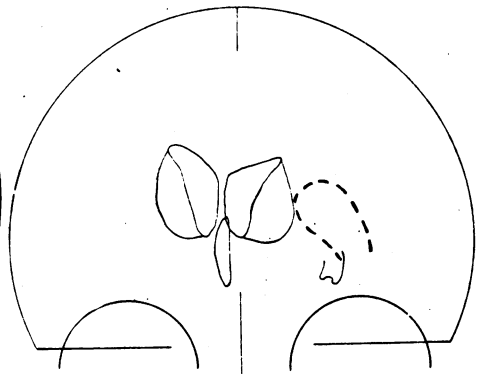


Fig. 25.

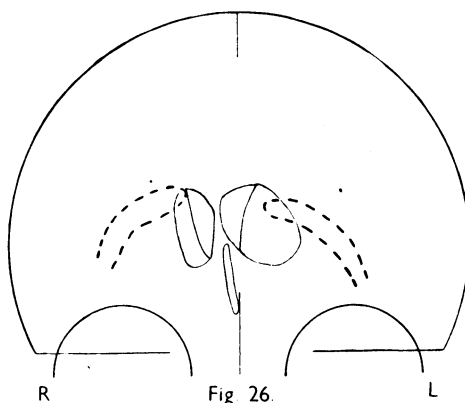
CASE 22.—Note medial and upward displacement of temporal horn.

most characteristic sign of the lesion. Although it cannot be called pathognomonic, for theoretically any expanding lesion of the same shape and position in the cranium could produce similar findings, in practice this must be very rare. Other expanding processes in this region will be the subject of a further research at the Röntgen

Department of the Royal Serafimer Hospital. But it is likely that a meningioma in the necessary situation is the only process which could simulate it, and had a meningioma grown so large it is highly likely that changes in the overlying bone or in the vessels would be present.

One such case was observed recently in which the pneumo-encephalographic features were typical of a subdural hæmatoma. There were no changes in the overlying skull, but the foramen spinosum on the affected side was much enlarged (fig. 21, Plate II). This indicated that the middle meningeal artery was hypertrophied, thus confirming the diagnosis of meningioma. An infiltrating growth such as a malignant glioma would be likely to produce localized deformities as well as general deformity, i.e. irregularity of contour of the lateral part of the affected ventricle, in contrast to the distant pressure effect of the subdural hæmatoma.

Of the remaining six cases which were air-filled, two (Nos. 10 and 19) showed filling of the contralateral and third ventricles only (fig. 22). These structures showed the features described above. Thus 14 out of 18 cases showed what I call the "classical picture".



CASE 9.—Bilateral hæmatoma. Note absence of classical characteristics. See text for description.

Of the remaining four cases, two (Nos. 1 and 17) showed almost normal appearances (fig. 23, Case 17), with just a slight displacement of the septum pellucidum. Both of these cases had small subdural hæmatomas.

One case (No. 22), with a hæmatoma in the base of the middle fossa, gave an atypical picture. Here a lateral drawing (fig. 24) is included to show the elevation and raggedness of the temporal horn. In the frontal view (fig. 25) the temporal horn is displaced medially and upwards. The ragged character of the temporal horn (lateral view) is difficult to explain, for it suggests an intra-cerebral lesion. This was in fact the radiological diagnosis, but at operation only a temporal subdural hæmatoma could be found. The patient died but no autopsy was obtainable.

One case (No. 9)¹ showed an atypical picture (fig. 26). This was not recognized at the time, and the condition was assumed to be merely a left-sided subdural hæmatoma—the diagnosis was made clinically and the encephalographic appearances seemed to confirm it. At operation a left-sided subdural hæmatoma was found. However, the patient did not recover consciousness after operation, repeated extradural hæmatomas formed and he was reoperated upon, but died, after three days, without recovering consciousness. At autopsy a subdural hæmatoma, 1 cm. thick,

¹ This case and Case 4 were described by Professor Ingvar and Dr. Ask-Upmark in their paper on subdural hæmatoma (*see* References).

was found on the other (right) side. Thus there are two bilateral cases in the series. In the other case the hæmatoma on each side was discovered on burring, preparatory to making a ventriculography. Bilateral hæmatoma should thus be borne in mind. On re-examining the encephalographic drawing of Case 9 in the light of the knowledge gained at autopsy one observes :—

(1) That neither temporal horn was filled. This might or might not have been of assistance, but if both were displaced medially, or if only the horn on the contralateral side was displaced medially, a bilateral lesion is certain. If the contralateral temporal horn was not displaced the diagnosis would be made more difficult.

(2) The drawing does not display some of the typical features shown in the 12 typical cases.

(a) There is no flattening of the roof of the "affected" lateral ventricle.

(b) There is no dilatation, but rather a diminution in size of the contralateral ventricle, and moreover the lateral border of its roof is not elevated but depressed.

(c) The third ventricle (anterior part) is practically vertical instead of being angulated.

In the light of these findings, and noting the contrasting appearances as compared with the typical cases, one should be very suspicious of the presence of a bilateral hæmatoma, in such a case.

Pneumo-encephalographic technique.—Lysholm's (1937) technique has been followed throughout. By utilizing his nine standard projections, and given sufficient air, it should be possible to outline the whole ventricular system and synthesize it by drawings. In practice this does not usually occur without special manipulation of the head, e.g. filling the temporal horn or fourth ventricle, and then taking subsequent pictures.

Technique for subdural hæmatomas.—Three pictures are taken in the supine position (Lysholm Ventriculogram Part I), and then examined. If a subdural hæmatoma, or in fact any expanding process in the lateral cerebral region, is suspected on the findings of these pictures, one then takes two pictures (one anteroposterior and one lateral) in the sitting position. By performing this manœuvre early in the examination a negligible quantity of air is lost from the ventricular system (the head is moved so that no flow of air through the aqueduct and exit foraminæ is allowed), and thus the cella media is examined.

The temporal horn of the affected side is next examined. It is very rarely filled on the first three pictures, so active manipulation of the head is necessary. This is quite simple—the head is merely turned so that the suspected temporal horn lies uppermost, and so air is allowed into it. The head is then replaced, the air being trapped in the temporal horn. The first three projections are then taken again. If the temporal horn has not been filled the process must be repeated. The contralateral temporal horn may then be filled by a similar manœuvre, as a control. This is only necessary in diagnosing very slight displacements of the temporal horn. The disadvantage of manipulating air into the contralateral horn is that some air is lost from the affected lateral ventricle, via the foramen of Monro, air which is required in examining the trigone of the affected side.

After examining the temporal horns one places the patient in the prone position and takes the remaining standard projections. It is very rarely necessary in investigating this particular lesion to take any more pictures.

Falx pressure.—Pressure by the falx on the trigonal region of the affected side is very commonly seen in these cases. It produces a quite characteristic deformity, and in the literature it is frequently interpreted as being due to pressure from an expanding process.

A reconsideration of the anatomy of the falx cerebri may assist in making the sign clearer. It will be remembered that the anterior part of the ventricle cannot come in contact with the falx at all, for the falx lies too high up and too far anteriorly.

Lateral pressure on the trigone, however, will cause it to come in contact with the falx almost immediately. It will then herniate under the falx and thus an impression is made on the trigonal outline (fig. 27). This sign is best seen in the projection VII (Lysholm) lateral projection—prone position, head not turned to right or left. If the posterior horns of the lateral ventricles are large, and if the quantity of air is not great, the air-fluid level may be too far posterior to see the falx impression. It is occasionally seen, however, with the head in the lateral position.

Fig. 28 is a reconstruction from a number of half-axial views of cases of subdural hæmatoma. It is semidiagrammatic and is intended further to clarify the condition. It is a view of the ventricular system from above in the classical type of subdural hæmatoma displacement. A cross section of the hæmatoma is shown on one side. The tentorium and the anterior borders of the middle fossæ are shown. The part of the falx which comes in contact with the ventricular system is shown, as well as the anterior portion. Note the slimness of the latter portion. The trigone is shown herniating under the falx. It also shows the relative dilatation of the contralateral ventricle, the curvature of the septum pellucidum, and the shift of the temporal horn on the affected side. In Case 2, when ventriculography was performed, some clear fluid was removed on puncturing the brain, and when the air was injected most of it was seen to be subdural, and outlining the falx (figs. 29, 30). This is the only case in which a complete outline of the falx has ever been observed at the Royal Serafimer Hospital.

Stereoscopy.—In the Lysholm technique stereoscopy is very rarely employed. Lysholm has found that by manipulating the head in various directions the whole ventricular system may be filled, and by taking pictures in two right-angled planes with the head in the various positions, localization of any deformity may be made. Half-axial views are also taken, for they provide an additional "dimension" and are of help occasionally in orientating deformities.

Finally, in every case a drawing is made of the ventricular system both in antero-posterior and lateral views. This provides a more accurate geographical plotting of the lesion than stereoscopy, which is subjective rather than objective.

Arteriography.—This is a useful ancillary method in the localization of intracranial expanding processes, and very often leads to a more accurate pathological-anatomical diagnosis than does pneumo-encephalography.

This investigation was performed on three of the cases. The first case (No. 8) was complicated by a pituitary tumour and only a lateral arteriogram and venogram were taken. No abnormality was discovered. In the other two cases (21 and 25) a malignant glioma was suspected on the history and clinical findings. The ventriculogram performed in each case showed the characteristic picture of a lateral expanding process (figs. 8, 11). Had the arteriogram shown an inoperable malignant glioma, the patient would have been saved an unnecessary operation.

Neither lateral arteriogram showed any abnormality, nor did the venograms.

The antero-posterior picture, however, in both cases showed a pathognomonic appearance both in the arteriograms and venograms (fig. 31). It will be seen that no contrast penetrated the hæmatoma, thus giving it a negative outline.

The arteriograms were taken towards the end of the injection of 8 c.c. of thorotrast—the venograms three seconds later. A stationary Lysholm grid was used incorporated in an apparatus designed by Lysholm-Schönander. This apparatus fits into the Lysholm-Schönander skull table and merely consists of a grid and a cassette holder for two cassettes, one above the other. The cassettes are separated by a tray of lead. The upper cassette and lead tray are quickly removed after taking the arteriogram. The second cassette is then in position for the taking of the venogram.

Post-operative air filling.—In two cases (Nos. 10, 13) air was injected into the

PLATE III.

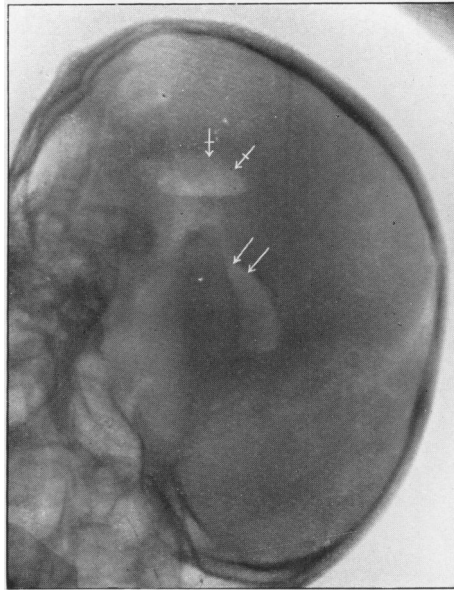


FIG. 27.—Trigone $\downarrow\downarrow$ Contralateral ventricle $\downarrow\downarrow$. Note similarity to fig. 20.

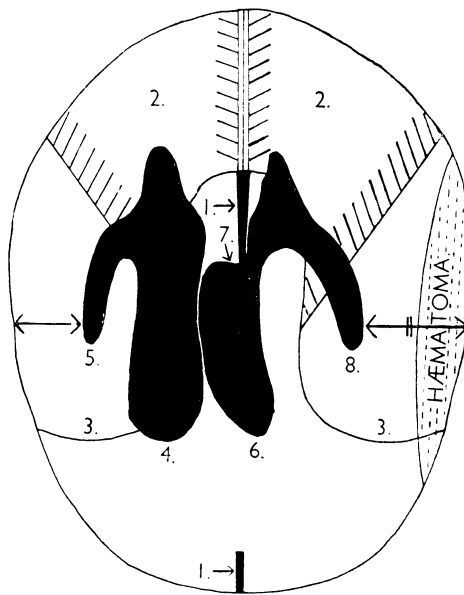


Fig. 28.

Semi-diagrammatic representation of ventricular system seen from above. Constructed from half-axial views.

- Key.—1. Falx in cross section. 2. Tentorium. 3. Anterior border of middle fossa. 4. Contralateral ventricle. 5. Contralateral temporal horn (normal position). 6. Homolateral ventricle. 7. Region of falx pressure and herniation under falx. 8. Affected temporal horn—shifted medially. Note difference in length of \longleftrightarrow and \longleftrightarrow .

PLATE IV.

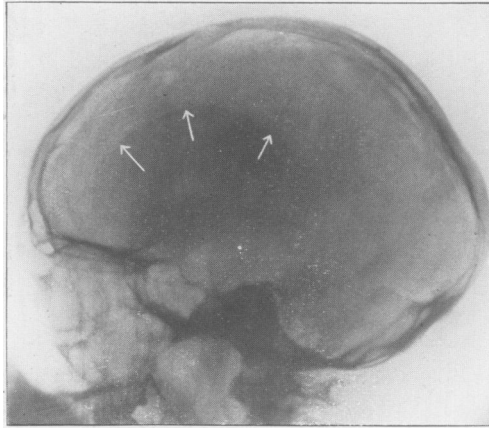


FIG. 29.—Air along falx and tentorium in subdural space.



FIG. 30.—Half-axial view of fig. 29.

PLATE V.

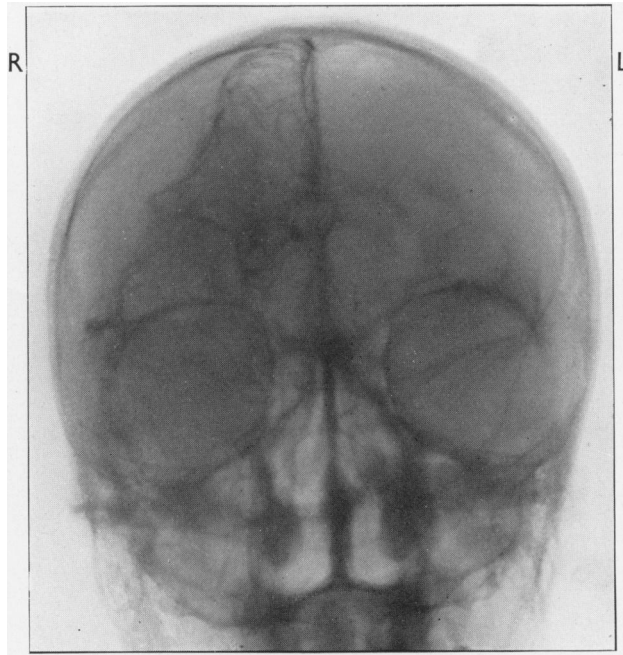


FIG. 31.—Arteriogram. Note absence of vessels at site of hæmatoma (right side).



FIG. 32.—Air replacement after operation. Note fibrous strand traversing hæmatoma cavity.

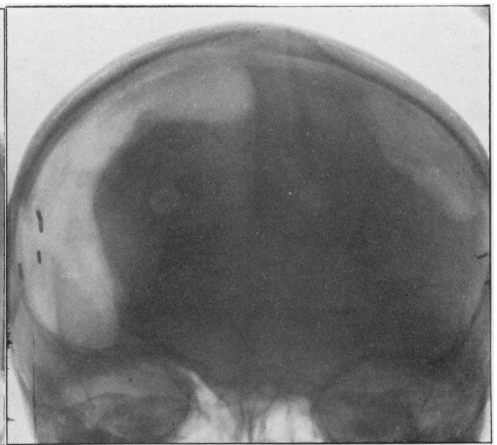


FIG. 33.—Air replacement after operation (bilateral case). Note silver clips at sites of drainage.

space left after evacuation of the hæmatoma. This was performed about ten days after operation.

Case 10 (fig. 32) shows an unusual feature. Running across the middle of the hæmatoma is a fibrous strand.

Case 13 (fig. 33) was one of the two bilateral cases in the series. The radiogram illustrates very well the extremely marked reduction in brain volume which occurs in such cases. Perhaps one should rather call it "brain anæmia", for probably the brain itself is not compressible.

DISCUSSION

The question at issue is whether or not there are constant and characteristic radiological findings in chronic subdural hæmatoma, and if one is agreed that there are, whether or not they are pathognomonic. Let us consider the straight X-rays first. It is generally agreed that fracture of the skull is extremely rare. Dyke and Davidoff found no skull fracture, nor was there any in the present series, and Kunkel and Dandy found fracture in only one case.

In the present series evidence of increased cranial pressure as shown by bony changes in the skull could be found in only two cases. Dyke and Davidoff, on the other hand, found atrophy of the sella turcica in 11 of their cases (45·8%). But they virtually contradict this later in their paper in discussing the pneumo-encephalographic findings, they say: "The entire ventricular system shows a marked shift to the contralateral side. In spite of this, the accompanying roentgenographic and clinical signs of *generalized increased intracranial pressure are minimal or absent*" (italics mine).

Kunkel and Dandy's findings are far more in keeping with the findings in the present series. Only two cases showed evidence of increased pressure in the straight X-rays, and one of these was an infant aged 9 months, whose skull would of course show signs of increased pressure long before that of an adult.

The position of the pineal gland is of importance. In this series it was visualized in eight cases (30%) and was shifted laterally in two (25%) of these eight cases. Dyke and Davidoff saw it in 15 cases (62%) and it was shifted laterally in five (33·3%) of these cases. They claim that in 12 cases it was displaced posteriorly from 0·1 cm. to 1·1 cm. These latter figures are of very doubtful value, particularly as Dyke and Davidoff do not state what standards they adopt, what technique they use, or what they regard as the normal "limits" of the position of the pineal gland in different shaped skulls. It is difficult to understand how a posterior shift of 1 mm. can be diagnosed as being pathological when the gland itself may calcify to a diameter of 1 cm., and the skulls vary so much in shape that displacement, on the lateral view, of 1 mm. surely cannot be stated with any confidence to be a displacement at all. As the hæmatoma is nearly always laterally placed and at a considerable distance from the pineal gland, backward displacement must be of very doubtful significance. But they state: "... the pineal displacement was relatively great, particularly in comparison to the slight signs of increased intracranial pressure".

Lilja's (1934) statistical survey of 200 cases showing calcification of the pineal, which presented no radiological or clinical evidence of an intracranial expanding lesion, illustrates this point. The normal variation of position was quite considerable.

I consider that in subdural hæmatomas the only significant finding associated with the pineal gland is lateral displacement, i.e. that seen in A-P or P-A projection. Calcification of the hæmatoma would appear to be the only pathognomonic sign on the straight X-rays.

Pneumo-encephalographic features.—Let us first consider hydrocephalus. There is no unanimity of opinion on this point. Kunkel and Dandy state: "Hydrocephalus

of varying degree was present in six out of nine cases . . . the cause of hydrocephalus was compression of the aqueduct of Sylvius." Dyke and Davidoff, on the other hand, say: "The lateral ventricles were visible in 11 out of the 15 cases and their size was well within normal limits, in fact, towards the lower limits of normal, with the exception of one case."

These two views are completely opposed to one another. In the present series all cases showed some degree of hydrocephalus and as shown (in figs. 13, 14, 15) the average was quite considerable.

The cause of the hydrocephalus is not convincingly clear. Kunkel and Dandy state (*see above*) that it is due to compression of the aqueduct of Sylvius. Is this always so? If the aqueduct is compressed it must almost certainly be displaced also. What is the evidence for such displacement? The aqueduct, lying deep in the brain-stem, is below the pineal gland. If the pineal gland, which lies at the back of the third ventricle, is not displaced laterally, the aqueduct is still less likely to be displaced. When the pineal was calcified in Dyke and Davidoff's series only 33.3% of these cases showed lateral shift and in the present series only 25%. It is therefore by no means conclusive that the aqueduct is compressed. As Ingvar and Ask-Upmark (1938) point out, the brain-stem may be likened to the root of a vegetable, and this root is much more firmly anchored at the base of the skull by vessels and nerves than the rest of the brain. This makes it even more unlikely for the aqueduct to be displaced by a process impinging on the convexity of the cerebral hemisphere. The fact that the upper portion of the anterior part of the third ventricle is so constantly displaced does not alter the argument; furthermore the bottom of the third ventricle (*see* figs. 1-12) is not displaced. The aqueduct is deeper in the brain than the anterior part of the third ventricle, and further away from the expanding process, and has even less chance of being displaced.

The most convincing explanation of the hydrocephalus appears to be that of Munro (1938) who holds that when the subdural space is distended a certain number of arachnoid villi are put out of action. Thus the production-absorption balance of cerebrospinal fluid is upset and hydrocephalus results. But even this theory is not altogether satisfactory, for in the one bilateral case in the series with air filling (fig. 26) there is an absence of hydrocephalus. With so many more arachnoid villi pressed upon by the hæmorrhage one would expect an even greater hydrocephalus.

Kunkel and Dandy state: "In three cases (33.3%) the ventricle was entirely collapsed on the affected side." But frequently on performing a ventriculogram or encephalogram one finds that the affected ventricle is not filled. This applies not only to subdural hæmatomas but to a variety of expanding processes in this region. But if on finding such a state of affairs one makes a burr hole in the frontal region on the affected side one finds that invariably the affected ventricle can be filled. At the Royal Serafimer Hospital, out of thousands of X-ray examinations and autopsies, the affected lateral ventricle has never been seen to be completely collapsed.

Again Kunkel and Dandy state that: "The reduction in the ventricular volume may be greater in front, greater behind or essentially equal throughout." While admitting that the ventricular volume on the affected side is less than that on the contralateral side owing to the lateral and downward pressure, and the falx pressure, this difference in volume is not very great. Also it was found with only one exception (Case 26) that the anterior borders of the two lateral ventricles were side by side. The posterior border cannot be assessed, for in the normal the occipital horns on either side vary very much. In Case 26 the anterior border of the affected lateral ventricle was displaced less than 1 cm. backwards.

CONCLUSIONS

The only pathognomonic radiological sign on the plain X-rays—that of calcification—was never seen. It has been observed in children and one case will be recorded in

another paper by the author, but it seems likely that adults die if untreated before the lesion has time to calcify, a process which probably takes years to come about. The pneumo-encephalograms showed constant features (excluding the one bilateral and the atypical cases).

Thus although no finding can be said to be absolutely pathognomonic, the circumstantial evidence is so great that the operative approach should be made with this diagnosis in mind, and a diagnostic burr hole drilled. In this way the reflection of a large osteoplastic flap may be avoided and the patient is submitted to a minor rather than a major operation.

A more difficult problem is the bilateral case. As stated above in describing the encephalographic changes in the one case of this type with air filling, certain atypical features are noted, and should make one suspicious. Other ancillary aids should perhaps be sought. Arteriography would not help unless injection was performed on both sides. This routine is adopted in very few clinics. It is possible, however, that electro-encephalography might be of assistance.

Arteriography: In the antero-posterior position it gives a pathognomonic sign as described above, but it is very doubtful if this investigation is justified in subdural hæmatoma, for although injection of the internal carotid artery is a small operation in itself, thorotrast is probably not a harmless drug.

I wish to express my thanks to my chief, Dr. Erik Lysholm, but for whose inspiration and help this work would not have been performed. I also wish to thank Professor Herbert Olivecrona for kindly placing his material at my disposal, and for his advice.

Finally, my thanks are due to Dr. Erik Lindgren and all other colleagues at the Royal Serafimer Hospital for the kind assistance which they were ever prepared to give me.

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REFERENCES

- ALLEN, A. M., DALY, B. B., and MOORE, M. (1935), *J. Nerv. & Ment. Dis.*, **82**, 193.
 BOWEN, W. H. (1905), *Guy's Hosp. Rep.*, **49**, 21.
 BURROWS, G. (1835), *Lond. Med. Gaz.*, **16**, 710.
 DYKE, C. G., and DAVIDOFF, L. M. (1938), *Bull. Neurol. Inst. New York*, **7**, 112.
 Id. (1938), *ibid.*, **7**, 95.
 HEWITT, PRESCOTT (1845), *Med. Chir. Trans.*, **28**, 45.
 HOLT, W. J., Jr., and PEARSON, G. B. (1937), *Arch. Neurol. & Psychiat.*, **37**, 1161.
 INGVAR, S., and ASK-UPMARK, E. (1938), *Acta med. Scandinav.*, **94**, 225.
 KUNKEL, P. A., and DANDY, W. E. (1939), *Arch. Surg.*, **38**, 24.
 LEARY, T. (1934), *J.A.M.A.*, **103**, 897.
 LILJA, B. (1934), *Acta Radiol.*, **15**, 659.
 LYSHOLM, E. (1931), *ibid.*, Suppl. XII.
 LYSHOLM, E., EBENIUS, B., and SAHLSTEDT, H. (1937), *ibid.*, Suppl. XXIV.
 MUNRO, D. (1934), *New England J. Med.*, **210**, 1145.
 Id. (1938), "Craneo-cerebral Injuries". Oxford.
 MUNRO, D., and MERRITT, H. H. (1936), *Arch. Neurol. & Psychiat.*, **35**, 64.
 SJÖQUIST, O. (1937), *Nord. med. tidskr.*, **13**, 711.
 TROTTER, W. (1914), *Brit. J. Surg.*, **2**, 284.

Discussion.—Dr. J. G. GREENFIELD said that Dr. Cumings and he had been investigating van den Bergh's reaction, as one of the ancillary methods of diagnosing subdural hæmatoma. Theoretically, if recent blood-clot was being absorbed and broken up, van den Bergh's reaction should be positive, and it was obtained in most cases of recent subdural hæmatoma.

A case in which bilateral calcified subdural membranes had formed presumably as the result of subdural hæmatoma, occurring during parturition or in infancy, was recently examined post mortem at the National Hospital, Queen Square. Fresh bleeding had occurred into the cavity between the calcified membrane and the skull on one side and led to the patient's death at the age of 16 years.

He agreed with Dr. Bull's summary of the pathology of subdural hæmatomata. It was difficult to understand why subdural hæmatomata continued to enlarge, sometimes, apparently, for months after the initial bleeding.

There were three theories for this continuous enlargement. Cushing and Putnam (*Arch. Surg.*, 1925, **11**, 329) said that the hæmatoma became covered with a membrane in which there were rather dilated thin-walled capillary vessels which continued to bleed into the substance of the hæmatoma for a long time under various provocations, with the consequence that fresh blood was always coming into the hæmatoma from this source. He was himself inclined to accept that view, because in every case examined fresh blood corpuscles were found in the hæmatoma, thus indicating recent hæmorrhage; and the only source from which it could come would be these dilated vessels.

The other two theories seemed more unlikely. One was that the broken-down blood in the hæmatoma, owing to its increase in osmotic pressure, absorbed cerebrospinal fluid or some other fluid from the neighbouring tissues. That would mean that eventually in the hæmatoma there would be a fluid containing probably low protein with a large amount of breakdown products of protein, which had not actually been found.

The third theory was that the hæmatoma was not really subdural at all but intradural, stripping off the so-called inner layer of the dura, which was very vascular, and therefore with a tendency to bleed. Those who had seen subdural hæmatomata either at operation or autopsy would agree that they could be stripped off the dura with the greatest ease, leaving a perfectly smooth dura behind. There was no evidence that the hæmatoma occurred in the layers of the dura, at least in the ordinary traumatic cases.

Major D. E. DENNY-BROWN said that the method of encephalography described by Dr. Bull sounded a very rational and successful procedure. It was remarkable that he got so little air in the subarachnoid space, and that presumably was why he had not seen the cleft appearance of subarachnoid air under the hæmatoma said to be characteristic of subdural hæmatoma by Dyke and Davidoff.

He had seen two calcified subdural hæmatomata. One of these was a case reported by Critchley and Meadows (*Proc. Roy. Soc. Med.*, 1932, **26**, 306 (Sect. Neur., 12)). The other case was in a woman aged 35 whose initial injury and onset of symptoms occurred at the age of 15. There were deformities of the skull like those which Dyke and Davidoff described in the sphenoidal fissure and the roof of the orbit, apparently only occurring as the result of the prolonged enlargement of the middle fossa by the subdural hæmatoma during the period of growth.

Perhaps he might add that he had come across a triple subdural hæmatoma. This was a patient in whom subdural hæmatoma was suspected, the usual burr holes were made, and bilateral subdural hæmatomata were drained with resulting recovery of consciousness. After about twelve hours deep stupor reappeared. Naturally it was suspected that there was obstruction of drainage, but investigation showed no such obstruction. The patient succumbed. At autopsy a very large occipital cyst, quite separated from the two parietal cysts by a partition, was shown to have been enlarged by a further collection of fresh blood. He was not aware of a description of a triple sac in the literature.

Dr. G. E. F. SUTTON said that Dr. Bull had mentioned another method of diagnosis, namely, the electro-encephalograph. He had recently seen a patient operated on by his colleague, Professor Rendle Short, on a diagnosis made by Professor Golla at the Burden Neurological Research Institute at Bristol. The diagnosis had been made by electro-encephalography, which showed, in contradistinction to the physical signs, exactly where it lay.

Mr. D. W. C. NORTHFIELD said he agreed that the clinical picture of subdural hæmatoma might vary widely, thus adding to the difficulty of making a correct diagnosis. The symptoms and signs might be only those of increased intracranial pressure due to a space-occupying lesion. The common problem was to decide between a malignant tumour, an abscess, and a subdural

hæmatoma, any of which might give rise to similar symptoms of a few months' duration and produce inconclusive localizing neurological signs or no such signs at all. On the other hand even outspoken focal neurological signs which would suggest a large intracerebral lesion might be produced by a hæmatoma. He described two cases illustrating this point.

At times the mode of onset and the distribution of the headache might be much more suggestive of subarachnoid than subdural bleeding, and the finding of a yellow cerebrospinal fluid would add to the difficulty of making a correct diagnosis. This was so in two other cases which Mr. Northfield described.

The other point he wished to raise in discussion was that of intracranial pressure. Headache was a constant complaint during some period of the history and was usually progressively severe and paroxysmal in type, denoting an expanding intracranial lesion. Although the hæmatoma might occupy a considerable amount of the intracranial space, yet objectively the intracranial pressure was seldom high and frequently abnormally low. In his own experience, papilloedema was not common. In only two cases of a recent series was the pressure of the cerebrospinal fluid abnormally high, and curiously enough in neither of these was papilloedema present. In one the lumbar pressure was 190 mm.; in the other the ventricular pressure was 600 mm., but the patient was congested from respiratory failure. In the remaining cases the pressure varied between zero and 150 mm. It would be argued that the lumbar pressures were not a true reading of the intracranial pressure owing to a partial block from herniation. This did not fully account for the findings because in some cases with a low lumbar puncture pressure, there was a good rise with jugular compression. The zero reading was of ventricular pressure, and in another case the ventricular pressure was such that fluid only trickled out slowly and a satisfactory measurement could not be obtained.

States of low intracranial pressure were frequently met with in cases of head injury, of long-standing hydrocephalus, and of tumours. It was perhaps most frequently seen in elderly persons with malignant tumours, and Dr. Greenfield suggested that this depended upon the greater roominess of the subarachnoid spaces which occurred in old age. Such an explanation was not applicable to the mechanics of all cases of subdural hæmatoma, for the low pressure was found in young as well as in old patients.

If W. J. Gardner's views (*Arch. Neur. and Psychiat.* (1932), **27**, 847) on the factor of osmosis in subdural hæmatomata were correct they offered a possible explanation of the low-pressure phenomenon. The greatest transference of water should occur through the inner and thinner membrane, the surface of which was applied to the brain. Water would thus be abstracted from the brain which would become compressed *pari passu* with the expansion of the cystic collection; thus the total bulk of the brain and cyst might not be increased.

Dehydration might be partly responsible for the low pressure in a few very ill patients, but could not be the cause in the majority.

Professor JEFFERSON said that Lysholm's reputation was so great that nothing need be said about it to such an audience, but it was pleasant to feel that one of their colleagues who had been sitting at Lysholm's feet had come back imbued with his methods and spirit and had been able to give such a paper and to show such pictures as Dr. Bull had done that afternoon. There was considerable doubt in the radiological world as to the uniformity, or even the possibility, of obtaining appearances of subdural hæmatomata by encephalography or ventriculography, particularly the first method which they used so much at Stockholm. Only a few years ago, when von Storch and Munro (*New Eng. Journ. Med.* (1938), **218**, 6) published a series of thirty-five cases they concluded that it was not possible to diagnose subdural hæmatoma by means of encephalography. This made Dr. Bull's present paper all the more important, because he had not only shown that it was almost uniformly possible to confirm the diagnosis in this way, but by a most interesting and careful dissection of the ventriculographic outlines he had shown, piece by piece, what was the meaning of the shift. He had made his case quite clearly that as a general principle only a tumour which was shaped like a hæmatoma and was situated where the hæmatoma was could fulfil all the requirements of the final picture.

Dr. J. W. D. BULL, in replying to Dr. Denny-Brown, said that an intra-hemispherical tumour such as a malignant glioma, would probably produce a localized deformity somewhere or other in the lateral wall of the affected lateral ventricle, without dislocation of the temporal horn.

He had been very interested in Dr. Denny-Brown's reference to the cases described by Dyke and Davidoff (1938) and two of his four infantile cases showed those appearances.

Dr. Bull thought that the disadvantage of using the electro-encephalogram only, was that it did not give the indication as to whether the lesion was extracerebral or intracerebral.

Mr. Northfield had raised the question of the clinical picture simulating a malignant glioma. It was for this reason that in Stockholm arteriograms were made on these occasions. Until they had realized the classical pneumo-encephalographic picture, it was the practice if a case was diagnosed as a malignant glioma, to make an arteriogram which gave the quite characteristic picture as described by Hemmingson (*Acta Radiologica* (1939), **20**, 499). It was very important to be quite certain that the lesion was not a malignant glioma because, as Mr. Northfield had pointed out, so many of these cases might be cured.